

*Clinical  
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GERTRUDE H. B. NICOLSON, M. D.

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## INTRODUCTION

Although varying in detail according to the individual case the average child's electrocardiogram follows a simple pattern which may be recognized as that of a normal child rather through its lack of abnormal signs than by the presence of any definite form other than that by which it is characterized as belonging to the childhood group.

In the following pages illustrative electrocardiograms of average children are shown together with tracings frequently found in certain abnormal conditions. It must be mentioned in this connection that the electrocardiogram may be of average form even in a badly damaged heart and it is to be remembered that the electrocardiogram is only an adjunct to a clinical examination.

The findings in this book are based on the study of electrocardiograms \* taken on 500 children from the wards and clinics of St. Luke's Hospital, New York City, and from private practice. For the sake of uniformity the tracings have been taken with the child in a sitting or propped up position. We have found that children do not fear the examination while in a sitting position as they often do when supine and again cardiacs in failure cannot lie flat during the test. Infant and child sized electrodes were used for smaller children.

The tracings of 350 physically normal fourth year students † between the ages of 22 and 26 years have been used as young adult tracings for comparison with this group.

The deflections and intervals present in a normal cardiac cycle are considered individually and as a whole. Normal variations are noted and abnormal tracings are described. Characteristics found in each

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## CHAPTER I

### *The Deflections and Internals of the Electrocardiogram of the Child*

**The P deflection** is the term given to the initial wave in the normal cardiac cycle which is the invasion or depolarization wave of atrial musculature during which time the impulse passes from the sinoatrial to the atrioventricular node. The time taken by the accession or invasion of atrial musculature is measured by the width of the P deflection. At birth and in the first weeks of life the average duration of this event is 0.056 second. The average pulse rate at this age is 164 per minute. There is a definite increase in P width as the child grows older as noted by the following measurements. From 2 to 12 months the

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0.078 second and the average rate is 112 and from 6 to 13 years the time consumed is 0.08 second while the average rate is 94 (Tables 1 and 7). In the young adult the average P width is 0.10 to 0.11 second.

The voltage or amplitude of the P wave varies in each lead and is greatest in lead 2 with an upper limit of 2 mm. In unipolar leads P waves are on the average highest in  $V_1$  and V

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may also be noted to a less marked degree in the tracings of the inactive rheumatic child (Fig 20) P waves may be absent as in atrial fibrillation (Figs 21a 25a and b and 57A)

The atrial T wave or wave of atrial repolarization may be recognized as a depression in the P R segment or it may fall after the QRS complex. The T<sub>a</sub> wave is

best seen in a v block of high degree and is apt to be more noticeable when P is high (Fig 56C 1 and 2)

The P R interval is the time taken for the impulse to pass from the s a node to the ventricular muscle The interval starts at the beginning of P and ends at the start of the R or of the Q wave if present It includes the time taken to depolarize the atrial muscle (P wave) plus the time in which the impulse is delayed at the a v node and the main bundle of His until the onset of ventricular depolarization (P R segment) The interval varies with heart rate and body size being shorter in more rapid and small hearts As would be expected the interval is shorter in the infant than in the young child and in the child than in the young adult The average time of the P R interval in the 1 to 8 week-old infant is 0.107 second with an average rate of 164 per

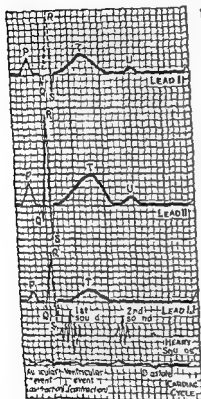


Fig. 1 Diagram showing events of the cardiac cycle by standard limb lead and standard wave cardiogram. Vertical squares = 1 mv. Horizontal squares = 0.04 second.

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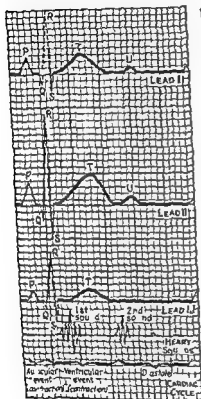


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during held inspiration there is an increase in the size of the R deflection in these leads (Patient referred by Dr Ruth Codmore)

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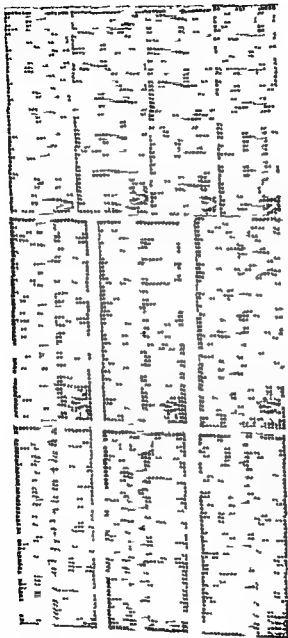


Fig 4 Electrocardiogram taken on 3 week old premature boy The voltage is low The heart rate is 160 There is slight elevation of the R T segment in lead 2

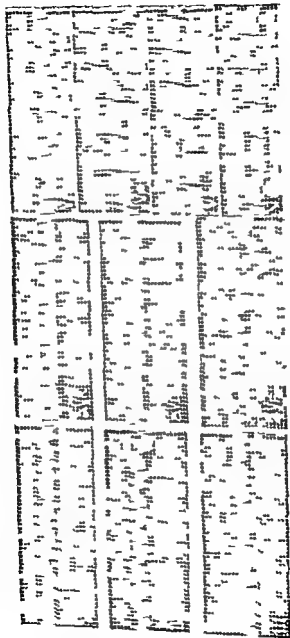


Fig 4 Electrocardiogram taken on 3 week old premature boy The voltage is low The heart rate is 160 There is slight elevation of the R-T segment in lead 2

Q or R to its end R or S. The average of 0.065 in the child is shorter than that in the young adult in whom the upper limit is 0.10 and occasionally 0.11 second. The QRS interval is usually shortest over  $V_2$  where the electrode is nearest the heart. The time varies with age, body size and build and heart rate being

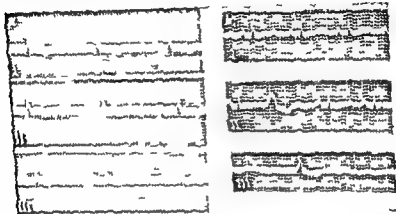


Fig 5 a. Marked low voltage in 17-year-old girl with untreated hypothyroidism. Note inversion of T waves in leads 1 and 2, slow heart rate and sluggish a-v conduction.

b. For comparison similar low voltage present in a 12-year-old girl with constrictive pericarditis. The rapid rate is the diastolic feature. Ventricular premature contractions present in leads 2 and 3 are coincidental.

greater in heavier hearts and shorter in infancy, in small hearts and in those with rapid rates.

The interval is commonly increased where there is a fibrosis of heart muscle, in cases where a bundle branch is blocked and in right and left ventricular hypertrophy. It may be transiently prolonged in acute infections as in acute rheumatic fever and during certain therapeutic procedures as under quinidization. In the Wolff-Parkinson-White syndrome the interval is lengthened.

The Q wave is the term given to the initial downward or negative deflection of the depolarization complex of ventricular musculature and is due to an initial potential difference directed away from the electrode.

In the normal child Q waves when present are larger than the

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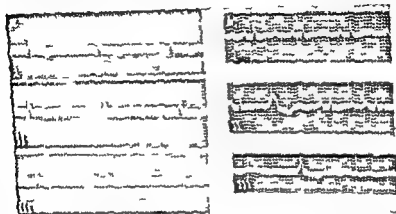


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muscle last to be activated such as the lateral and posterior basal wall of the left ventricle and are absent in tracings taken over muscle initially invaded or first to be activated as found over the right precordium. The child's Q wave is usually absent over  $V_1$  and  $V_2$  and is deepest in leads 3 and  $V_6$ . It is normally narrow with an average width of 0.0175 second and depth of 0.036 mm with a maximum of 7.5 mm.

Q waves are frequently seen in lead 1 and in precordial leads facing the left ventricle in left ventricular hypertrophy. They are common in lead 3 and precordial leads over the right ventricle in right ventricular hypertrophy.

In left bundle branch block Q waves are absent in tracings with the electrode facing the left precordium, a distinguishing point from those of left ventricular hypertrophy.

Abnormal Q deflections are usually wide and greater than 25 per cent of the highest R deflection in the lead.

The Q T interval starts at the beginning of Q and terminates at the end of T. It includes the time taken by the impulse to invade the ventricular muscle and the time for the complete return of the muscle to its resting or polarized state.

The interval varies with heart rate, sex, and age. It varies also with the electrolyte and water balance of the blood, as with potassium loss and sodium retention, with calcium and phosphorus changes, or disturbances of carbohydrate metabolism, androgenic and other factors. The interval is increased in heart failure, in myocarditis, and in ventricular hypertrophy during toxic states, in quinidine intoxication, and in avitaminosis, as well as in hypopotassemia and hypocalcemia. It is shortened in hypercalcemia and with digitalis effect, and occasionally in acute pericarditis. The lability of the interval owing to its response to such external factors makes it unreliable as a constant measure of precision. However, although it is prolonged in most infectious diseases including rheumatic fever, it is useful in determining the presence of activity in questionable rheumatic infection when serial tracings are made (Figs 17A, B, and C).

The average measured Q T interval together with its related average R R cycle may be converted to a uniform standard through

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its peak is shorter in leads from the right precordium than in those taken over the left precordium of the normal child. There is a slight increase in this time interval or delay in the intrinsicoid deflections as the child grows older but the relative ratio of right to left continues (Table 4). The peak of R divides the QRS deflections due to muscle activated before from that depolarized after the impulse reaches the muscle under the exploring electrode. There may be two upward waves in the QRS complex separated by a downward deflection. The second wave  $R^1$ , is often seen when the electrode is over the base of the heart from late activation of the conus. If slurring is present in the highest R near its apex, abnormal conduction is probable. The wave is commonly extremely high in congenital heart disease (Fig 13a). A late R peak in  $V_1$  with early intrinsicoid deflection in  $V_4$  is seen in right ventricular hypertrophy and right bundle branch block (Figs 13a and 46).

**S wave** is the designation of the final downward wave of the depolarization complex of ventricular muscle owing to negative cavity potentials being transmitted through a completely depolarized wall to the electrode over its epicardial surface.

The average depth of the S deflection in the standard limb leads of childhood is 1.61 mm and over precordial leads 6.66 mm. It is deepest in leads 2 and V and in the younger age group. It is often absent in the child and may vary in depth in I 3 during respiration (Figs 2 and 3).

S waves are deepest in tracings from that portion of the wall where activation is completed first as is usual in unipolar leads taken with the terminal over the right ventricle and absent where it is completed last as over the left ventricle.

**The junction or J'** is the term used for the meeting point of the QRS complex with the S-T segment.

**The S-T segment** is represented by the duration of time between the end of the S wave and the start of the T deflection. It is the interval of time between the completion of depolarization and beginning of repolarization of ventricular musculature. This segment varies according to the heart rate being longer in slow rates and shorter when the rate is rapid. The average duration of the S-T

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is also opposite to that of the T deflection. T wave changes from involvement of epicardial and endocardial ventricular surfaces may best be seen in unipolar limb leads.

*U wave* is the term given to the positive after potential of muscle contraction. It starts at the end of the T wave and is usually not visible in the child's tracing. However, in slow rhythms and in the presence of hypokalaemia it may occasionally be fairly prominent (Figs 53c and 55C).

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Fig 7 Standard and unipolar limb leads of children with average pre  
coral leads showing  
a 6 yr  
b 1 in 11 year-old girl  
c  
d  
e

Change in heart position with attendant variations in the relative relationship between the right and left ventricles may be found in the same child. During deep h/d inspiration for example the diaphragm descends and the heart being relatively fixed at its base will in following rotate on its longitudinal axis in a clockwise direction so that the left ventricle lies below the right ventricle

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Fig 7 Standard and unipolar limb leads of children with average precoral leads showing

- a 11 year-old girl
- 1
- 2
- 3
- 4
- 5
- 6

Change in heart position with attendant variations in the relative relationship between the right and left ventricles may be found in the same child. During deep tidal inspiration for example the diaphragm descends and the heart being relatively fixed at its base will in following rotate on its longitudinal axis in a clockwise direction so that the left ventricle lies below the right ventricle

deviation may depend on a mechanical shift in the relationship of the two ventricles to the chest or it may be due to disease or malformation in which hypertrophy of either ventricle is commonly present

The electrocardiogram of the normal child is peculiar to that child. Apart from the changes consistent with growth and from those variations seen in the axis deviation of the ventricular curves on rotation of the heart around its longitudinal axis the general characteristics present in a normal child's electrocardiogram are retained throughout childhood years with remarkable constancy.

Such differentiating points appearing to be inherent qualities of the individual's cardiac activity are inverted or diphasic  $\text{R}$  waves in leads 2 or 3 the presence of U waves the slurring or splitting of QRS complexes the origin of the S-T segments and the ratio of the voltage of  $\text{L}_1$  to  $\text{L}_3$ .

Many of these signs are due to the constitutional type of the individual child, and they may be found in several members of one family. Similarities in the tracings of siblings are often so striking that differentiation of the electrocardiograms is rendered difficult as in the records of the siblings illustrated (Figs 8 and 10). In Fig 9 the tracings of two children a brother and sister are compared. A resemblance is again present and shows low voltage of lead 1 in both and a diphasic  $\text{T}_3$  in one and inverted  $\text{T}_3$  in the other. Higher P wave amplitude is present in the girl and greater T wave voltage in the boy as is usually seen in comparison between the sexes.

The P wave of atrial activity is greater in height in the standard limb leads of girls between the ages of 6 and 12 years than it is in boys of that age group. The components of the QRST group of the standard limb leads are of greater amplitude in boys than in girls in the 6-to-12 age group (Fig 9). In this connection it is of interest to note that Beneke in 1910 observed in his studies on the anatomy of the heart that the relative volume of the atria to the ventricles was greater in the female than in the male.

At 12 years of age all standard limb lead deflections are on the average greater in amplitude in boys than in girls. The average heart rate in girls of 6 years and under and of 12 years and over

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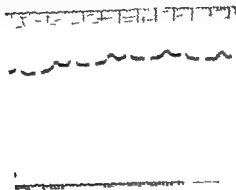


Fig III Tracing showing leads 2 from two brothers (A and B) 5 and 7 years old who had congenital heart lesions. Incomplete a-v block is present in both boys

standard lead electrocardiogram and the changing electrical field of the heart

For this study a record is made in the form of a loop by the junction of the apices of several instantaneous axes which are determined from the tracings of two or three simultaneous standard leads and noted at consecutive periods. Although time-consuming with present methods the vectorcardiogram promises to be a valuable asset especially in the study of congenital heart lesions

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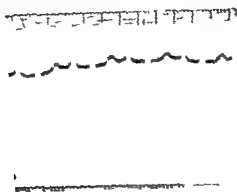


Fig 10 Tracing showing leads 2 from two brothers (A and B) 5 and 7 years old who had congenital heart lesions. Incomplete a-v block is present in both boys

standard lead electrocardiogram and the changing electrical field of the heart

For this study a record is made in the form of a loop by the junction of the apices of several instantaneous axes which are determined from the tracings of two or three simultaneous standard leads and noted at consecutive periods. Although time-consuming with present methods the vectorcardiogram promises to be a valuable asset especially in the study of congenital heart lesions.

**Standard lead 2** Electrodes are placed on the right arm and left leg so that potential variations from the right arm and left leg roots are noted in the tracing (When considering simultaneous points of the curves in different leads lead 2 equals the sum of leads 1 and 3 for all practical purposes) P waves are usually upright and greatest in this lead They may show a slurring on the upstroke and rarely they may be inverted as a positional effect Q waves are often absent but they may be deep R waves are usually tall When present S deflections are often deepest in lead 2 T waves also reach their greatest standard lead height in this lead very rarely they may be inverted because of heart position

**Standard lead 3** With electrodes placed on the left arm and left leg potential variations from the left arm and left leg roots are registered in the tracing P waves are inverted in lead 3 in about 10 per cent of normal children Upright R waves usually dominate the QRS complex in the normal child in this lead however in a horizontal electrocardiographic position left axis deviation with deep S or QS waves may be seen Q and S waves are often deep and may disappear or vary with position T waves are inverted in lead 3 in about 35 per cent of normal children

In contrast to these bipolar leads unipolar leads record potentials from one extremity only the exploring electrode the other the indifferent electrode being at the so-called zero potential As the deflections in unipolar limb leads are small augmented unipolar limb leads as introduced by Goldberger are almost universally used These are designated as  $aV_R$ ,  $aV_L$  and  $aV_F$  leads Unipolar leads are valuable as a means of comparison between the right and left ventricles The electrocardiographic curve varies according to the portion of the heart that faces the limb root on which the electrode is placed This may be roughly either the great orifices of the heart or the right or left ventricle In the normal heart a relationship exists between standard and unipolar limb leads so that if L1 resembles L2  $\sqrt{R}$  is similar but inverted and if L3 is the inverse of L1 VL resembles L1 also if L2 and L3 are similar  $\sqrt{F}$  is also similar Again  $VL - VR = L1$   $VF - \sqrt{R} = L2$   $VF - VL = L3$  (Fig 7)

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but may be diphasic or rarely inverted. The usual QRS complex in the VL lead of a child consists of an RS curve often of low voltage. However, the form depends on the electrical position of the heart and it is occasionally difficult to determine whether the base of the heart or the epicardial surface contributes more to the form of the QRS complex in this lead. If the electrocardiographic position is vertical the left arm root will face the clockwise rotated right ventricle and the tracing will show an rS complex and inverted T wave. If in a horizontal electrocardiographic position the left shoulder will face the counterclockwise rotated left ventricle and a qR will be inscribed and the P and T waves may be inverted when they will also be so in VR leads. The semivertical electrocardiographic position shows low voltage of QRS in VL and the semihorizontal electrocardiographic position gives low voltage in VF. The S-T segment, which is usually isoelectric, is occasionally elevated or depressed (Fig. 7).

IF, the unipolar left leg lead or  $aV_F$ , the augmented unipolar left leg lead, shows the potential variations of the left leg or left hip joint similar to those of the surface of the diaphragm facing the heart. P waves are usually upright in VF leads. The form of QRS in the child's VF lead depends upon the electrical position and rotation of the heart. In the vertical electrocardiographic position VF is similar to leads over the left ventricle and will show Rs or qRs ventricular complexes with high R waves. In the horizontal electrocardiographic position the QRS form in VF resembles those over the right ventricle showing deep S waves which may disappear on deep inspiration (Fig. 2). When the heart is in the semihorizontal position the QRS complexes in VF are small. When there is forward rotation of the apex the left leg lead will face the epicardial surface of the left ventricle and a qR will be seen in VF and when there is backward rotation an rS or Rs will be present reflecting potentials from the right ventricle.

Q waves in VF may be seen in normal tracings. They are usually followed by tall R waves with a late peak. A broad Q with small narrow R is abnormal as is true also when it is not accompanied by a QR in VR. Rarely T waves may be inverted normally in VF but they are usually positive in this lead.

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## CHAPTER IV

### *Unipolar Precordial Leads*

***Precordial Unipolar Leads*** According to the criteria of the New York Heart Association precordial leads are taken on the chest at desired points over a line extending from the right sternal margin at the level of the fourth intercostal space to the junction of the left midclavicular line with the fifth intercostal space continuing at this level around the left chest. Right-chest leads are taken over the right chest in a similar manner (Fig 11A and B)

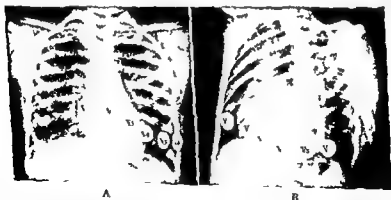


Fig 11 Positions of unipolar precordial leads shown by pennies on chest of 11 year-old asthmatic girl. A. Posterior anterior view. B. Left anterior oblique view. (After Hecht.)

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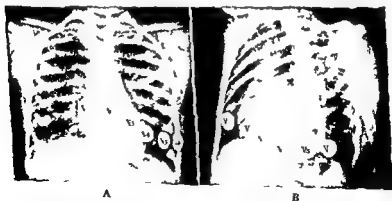


Fig 11 Positions of unipolar precordial leads shown by pennies on chest of 6 year-old asthmatic girl. A. Posterior anterior view B. Left anterior oblique view (After Hecht)



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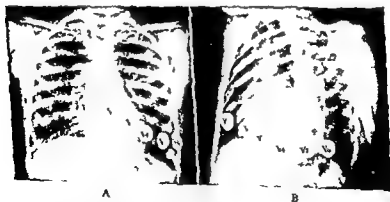


Fig 11 Positions of unipolar precordial leads shown by pennies on chest of 6 year-old asthmatic girl A Posterior anterior view B Left anterior oblique view (After Hecht.)

Illustration showing average pre intrinsicoid deflections with normal ranges in V<sub>1</sub> and in V<sub>6</sub> at different age groups of normal children

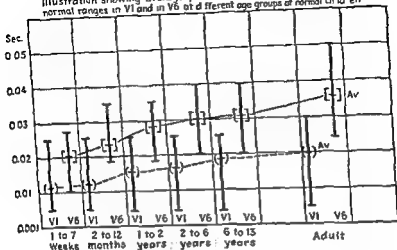


Table 4

diphase in the infant becoming less commonly so in the older child where they are usually clearly visible and with greater amplitude than V in other chest leads. In the newborn and the infant under 8 weeks an isolated R wave or R<sub>s</sub> curve followed by an inverted T wave is the rule for the QRS form in V<sub>1</sub>. The amplitude of the R deflection in V<sub>1</sub> gradually decreases with the age of the child so that in the 6- to 13 year old age group an rS is the usual form although the T waves continue to be inverted. Very rarely the R waves are absent and a deep QS is present. The intrinsicoid deflection occurs early in the course of the QRS group (Table 4). In the normal child T in V<sub>1</sub> is rarely upright and the ST junction is frequently elevated.

The various forms of the QRS complex found in V<sub>1</sub> are attributable in most part to the different positions in which the heart may be found. If the electrode should lie over the right ventricle the initial deflection will be an R wave. If it lies over the right atrium and faces the great orifices of the heart as it does when the heart is displaced backward and to the left, a QS or rS will be present. In this case R is prominent in aV<sub>R</sub> also and the transition zone will be shifted to the left.

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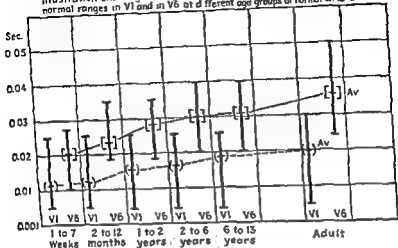


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**I<sub>1</sub>** (The tracing is taken with the exploring electrode placed at the left costosternal margin at the level of the fourth intercostal space ) P waves are occasionally inverted or diphasic in this lead in the child. The usual QRS form is an RS curve. Rarely a small q wave is seen in infants under 1 year of age. S waves are usually deepest in this lead although in the younger age groups especially S may be deeper in leads V<sub>3</sub> and V<sub>4</sub> than in V<sub>1</sub>. T waves are commonly inverted in V<sub>1</sub> in the younger child. They are inverted in 64.3 per cent of normal children under 6 years and 27.2 per cent in those of 6 to 13 years (Table 4). The S-T junction is usually isoelectric although it may be highly elevated or depressed in this lead (Fig 12).

**I<sub>2</sub>** (The exploring electrode is placed midway between the positions for V<sub>1</sub> and V<sub>4</sub>.) Because of the small size of the child's chest this lead and that of V<sub>3</sub> are frequently omitted from the child's electrocardiogram except in special instances. The initial deflection in V<sub>2</sub> is usually an R wave and T waves are usually inverted in children under 6 years of age.

Incidence of inverted and diphasic T waves in precordial leads in 135 healthy children at different ages.

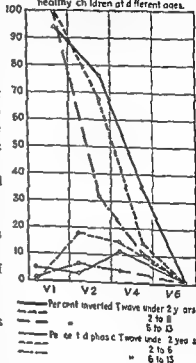


Table 5

As the age of the child increases, V<sub>1</sub> shows a greater amplitude of the R wave.

S waves are usually deeper in V<sub>1</sub> than in V<sub>2</sub>.

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The S-T junction is usually isoelectric although it may be highly elevated or depressed in this lead (Fig 12).

Small q waves are common after the second year. An R curve is usual at all ages in this lead.

R and T deflections are usually seen in V<sub>2</sub> and V<sub>3</sub>.

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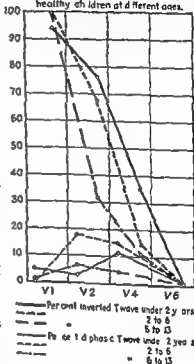


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As the age of the child increases, V shows a greater amplitude of the R wave.

S waves are usually

deeper in V<sub>1</sub> and V<sub>2</sub>

than in V<sub>3</sub> and V<sub>4</sub>

and high in the second year

upright. Small q waves

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and T deflections are

usual.

## *The Child's Electrocardiogram in Disease*

### **Bundle Branch Block**

Bundle branch block is due to an obstructing defect or anomaly in the conduction pathway of one or the other of the branches of the bundle of His resulting in delay in activation of the affected ventricle. Absent Q waves and broad notched or bifid R waves with delayed intrinsicoid deflections and widened QRS complexes of 0.10 second or more characterize the tracings over the epicardial surface of the blocked ventricle of the child. Narrow R deflections and broad S waves with widened QRS intervals are present over the opposite ventricle (Table 6 and Figs. 13a and b, 30, 35 and 41A).

Bundle branch block is not rare in congenital heart disease especially in lesions of the atrial septum and less commonly of the ventricular septum. It may be found in long standing cases of aortic regurgitation and mitral stenosis.

As a transitory finding bundle branch block may be seen in patients with acute infections such as diphtheritic myocarditis and acute rheumatic carditis. In the early stages of the chronic form transient bundle branch block may be noted. It may be seen as a manifestation of toxicity during drug therapy such as with quinidinization.

*Art orientation block* is the term occasionally used in describing bundle branch block which shows low voltage in the standard limb leads (Fig. 14).

(Text continued on p. 36)

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(Text continued on p 36)





Fig 14 Arborization block (bundle branch block with low volt age) shown in standard limb leads of 10-year-old girl with clinical signs of ventricular septal defect

#### Right BBB

NOTCHING OF QRS COMPLEXES WITH QRS INTERVAL  $\geq 0.10$  SECOND OR MORE

Form of QRS curve in limb leads depends on heart position. It is usual to see late wide M in leads I and VL, late notched R in VR with R in leads 3 and VF.

QRS complexes in V and V usually show rSR curves with wide and tall R and inverted T waves.

QRS in V and V usually shows qRS with prominent S and upright T.

The intrinsicoid deflection is delayed over the right precordium starting 0.02 second or more after the onset of the QR curve and is normal over the left ventricle.

#### Left BBB

NOTCHING OF QRS COMPLEXES WITH QRS INTERVAL  $\geq 0.10$  SECOND OR MORE

The QRS outline in limb leads depends on the heart position but usually shows high R waves in leads I and VL with deep S or QS in leads 3, VF and VR.

QRS in V and V usually shows QS or rS with elevated M T segment and upright T.

QRS in V and V usually shows absent Q waves, notched R waves with depressed M T segment and inverted T.

The intrinsicoid deflection is late over the left precordium starting 0.04 second or more after the QRS onset and is normal over the right ventricle.

Table 6 Differentiation between right and left bundle branch block in the child



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sinus or vagal stimulation. The P J segment will then be seen to be equal to the P J segment found during a cycle of ectopic rhythm. This is not the case in true bundle branch block, where the blocked P J segment is increased in duration.

### Cardiac Hypertrophy

Cardiac hypertrophy is usually first localized to a single heart chamber. There is an increase in the size and weight of each muscle fiber in the muscle mass affected. Other chambers may or may not be involved later in the course of the disease.

*Atrial hypertrophy* may be found in patients in whom an increased load is present in the atria of the heart as in those with pulmonic tricuspid or mitral stenosis, atrial septal defects, pulmonary disease, and certain types of myocardial infarction.

In such cases where there is marked atrial activity, the P waves may be wider than 0.11 second and as high as 2.5 mm. They may be peaked, tall, broad, notched, or slurred and are best seen in V<sub>1</sub>, V<sub>4</sub>R, and VL leads (Fig. 56A and C).

*Hypertrophy of either ventricle* commonly shows a delay in the duration of the intrinsicoid deflection in the tracing taken over the hypertrophied ventricle in comparison with that taken over the normal ventricle. The R waves are usually markedly increased in voltage, and small S deflections with depressed S-T segments may be seen in leads facing the hypertrophied ventricle, while the inverse is found over the normal ventricle. The QRS interval is moderately increased to 0.1 or 0.11 second over an hypertrophied left ventricle, and the transitional zone will be noted to be nearer the affected ventricle than normal.

*Left ventricular hypertrophy*, as may commonly be found in such conditions as tricuspid atresia and aortic stenosis, marked coarctation of the aorta, and long standing severe patency of the ductus arteriosus, may show the following changes:

I, and leads facing the right ventricle show rS or QS waves with no delay in the intrinsicoid deflection. The S-T segment is elevated in these tracings, and T is upright.

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intervals and right axis deviation. However, with the heart in a horizontal electrocardiographic position, left axis deviation may be found and occasionally in this position an rS curve may be seen in all standard limb leads. QR complexes are the rule in VR leads and Q waves with T inversion are common in leads 2, 3 and VF (Figs 36A and 48).

*Differentiation of right ventricular hypertrophy from right bundle branch block* is difficult at times. The shorter QRS interval and absence of notching of the R waves over the hypertrophied ventricle together with the presence of small R waves over the left precordium suggest hypertrophy rather than right bundle branch block (Figs 13a, 36A and 46).

### Ventricular Strain

Ventricular strain\* has been described as a functional disturbance of heart muscle which is usually reversible and whose electrocardiographic signs are similar to those of muscle injury which affects the repolarization process only. S-T segments become depressed and T waves inverted while the QRS curves remain unchanged. It has been suggested that when the S-T-T changes as noted above are found without increase in amplitude of R and S waves, ventricular strain without hypertrophy may be presupposed and that when the S-T-T changes described above are not found in the presence of increased amplitude of R and S deflections, hypertrophy without ventricular strain is present also. That combined QRS, S-T and T changes suggest the presence of hypertrophy with strain.

### Ventricular Myocardial Infarction

Ventricular myocardial infarction follows an acute ischemia of a part of ventricular muscle whose blood oxygen supply is either suddenly cut off or becomes inadequate for its nutritional demands. The affected muscle becomes necrotic and is replaced by fibrous tissue. The lesion is rare in the child and the diagnosis may be easily missed. Coronary circulation in the child differs from that of the adult. In the

\*There has been criticism of the use of the term "ventricular strain" in electrocardiographic interpretation.

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Ventricular strain\* has been described as a functional disturbance of heart muscle which is usually reversible and whose electrocardiographic signs are similar to those of muscle injury which affects the repolarization process only. S-T segments become depressed and T waves inverted while the QRS curves remain unchanged. It has been suggested that when the S-T-T changes as noted above are found without increase in amplitude of R and S waves, ventricular strain without hypertrophy may be presupposed and that when the S-T-T changes described above are not found in the presence of increased amplitude of R and S deflections, hypertrophy without ventricular strain is present. Also that combined QRS, S-T and T changes suggest the presence of hypertrophy with strain.

### Ventricular Myocardial Infarction

Ventricular myocardial infarction follows an acute ischemia of a part of ventricular muscle whose blood oxygen supply is either suddenly cut off or becomes inadequate for its nutritional demands. The affected muscle becomes necrotic and is replaced by fibrous tissue. The lesion is rare in the child and the diagnosis may be easily missed. Coronary circulation in the child differs from that of the adult. In the

\*There has been criticism of the use of the term "ventricular strain" in electrocardiographic interpretation.

intervals and right axis deviation. However with the heart in a horizontal electrocardiographic position left axis deviation may be found and occasionally in this position an rS curve may be seen in all standard limb leads. QR complexes are the rule in VR leads and Q waves with T inversion are common in leads 2, 3 and VF (Figs 36A and 48).

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The lesion is transmural and is most frequently found in the anterior and apical portions of the left ventricle which is the area supplied by the anterior descending branch of the left coronary artery the vessel most usually affected

Occlusion of the right coronary artery will cause infarction of the posterior wall of the left ventricle and often includes the posterior portion of the septum in the affected area

The electrocardiographic diagnosis is made only on characteristic changes in the QRS complexes with S T displacements and, or typical T wave changes

The first electrocardiographic sign of myocardial infarction which may be found is typical of muscle injury and is represented by an upward displacement of the RS T segment over the affected area. The elevation is usually transitory and of short duration and it disappears when the affected muscle dies or recovers

The voltage of the positive intrinsic deflection decreases and may disappear while a deep negative or Q wave appears over the necrosed area this being incapable of impulse response allows the potential variations of the adjacent ventricular cavity to be transmitted through the infarction to the overlying electrode. If portions of living muscle are present in the lesion small R waves or notching of the Q waves will be seen. These signs may be transient or permanent

T waves registered from marginal epicardial portions of the necrosed area may become deeply inverted in their terminal portions and may remain so indefinitely

When electrocardiographic signs of myocardial infarction are present progressive changes are early and rapid in serial tracings

An anteroapical wall infarction of the left ventricle may show typical changes in LI VL V<sub>1</sub> V<sub>2</sub> and V<sub>3</sub>. In anterolateral wall infarction disturbances may be seen as well in leads V<sub>4</sub> and V<sub>6</sub>.

and depressed and inverted T waves. The S-T segment is depressed in V<sub>1</sub> and elevated in V<sub>4</sub>. Interpolated and ectopic auricular beats are present in leads I and in aV<sub>1</sub>

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nomonic of the condition those showing deep S waves and persistent displacement of RST segments in leads 2 and 3 together with high R and upright T waves in VR and those having deep or QS deflections with inverted T waves in lead I and high R wave in lead 3. Unusual rotations of the heart following myocardial injury are the main causes of these electrocardiographic changes.

### Rheumatic Fever

Transitory electrocardiographic changes are usually present to some degree in acute rheumatic carditis.

During my internship and later while attending on the Children's Service at Bellevue Hospital in the 1920's I studied a group of 100 children of 13 years and under who had active rheumatic carditis and to whom digitalis was given only rarely and sparingly at the time. Electrocardiograms were also then repeated only occasionally but each child in the group had at least one tracing taken. Of the 61 showed first-degree a-v block, 11 second-degree block while 4 children complete a-v dissociation was present. Atrial fibrillation occurred in 9 cases of whom the youngest was 9 years old (7 of the children died within a few months after the onset of fibrillation). 1 child showing atrial fibrillation combined with complete a-v block and 1 ventricular fibrillation as a terminal event.

The findings depend upon the severity and location of the lesion. If these are diffuse abnormalities may be found in all parts of the cardiac cycle with P wave and QRS curve widening and notable S-T displacements and T wave changes. Disturbances of impulse conduction in the uncharted pathways of the atria or in the bundle of His and its branches are recognized by a widening of the P-R or QRS intervals when in the a-v node the bundle or its branches. When there is delay in the invasion and retreat of the impulse through inflammatory reaction of the ventricular muscle the segment shows an increase in time duration. If the epicardial surface of the heart is affected as in acute pericarditis S-T displacement

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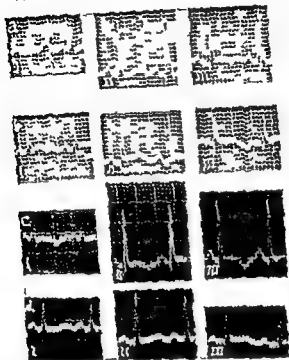


Fig 17A Standard limb leads taken over consecutive weeks on a 12 year

c The T deflections have become deeply inverted in all leads and the ST segments are isoelectric but *co. line to show upward convexity* and the QT of 0.407 second is again above normal

d The tracing returns to original form. The QT continues to be prolonged at 0.403 second

### Acute Rheumatic Pericarditis

Acute pericarditis frequently shows a typical electrocardiogram in the early stages of the acute process. The changes are usually rapid and transitory depending however on the nature of the infection. They are characteristic of *epicardial injury* and affect first the deviation of the ST segment and later the T deflection. The first sign is a noticeable elevation of the S-T segment in the three standard limb leads

(Text continued on p 48)

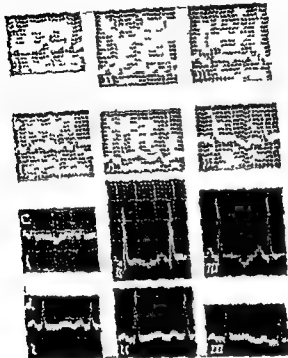


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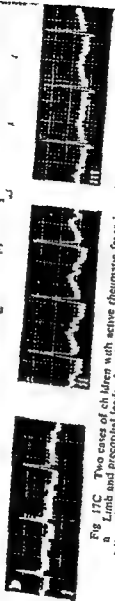


Fig 17C Two cases of children with active rheumatic fever in early stage  
 a Limb and precordial leads of an 8 year-old boy showing rapid sinus rhythm and sluggish  
 a v conduction. The P R interval is 0.21 second  
 b Standard limb leads of an 8 year-old boy with inflamed and tender joints. The  
 P R interval is 0.21 second and the R T segments in leads 2 and 3 are elevated. The P waves  
 are wide and the P R segment is depressed in leads 1 and 2



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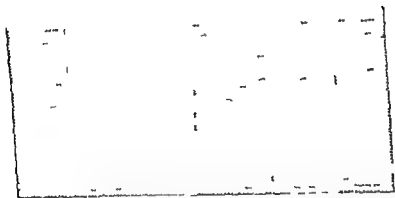


Fig. 19. Standard and unipolar limb leads and unipolar precordial leads from a 16 year-old boy with inactive rheumatic aortic disease and hypertension. The tracing shows sinus rhythm with marked left axis deviation and early left ventricular hypertrophy. The deep S present in lead 3 does not change on deep inspiration in cases of left ventricular hypertrophy.

hydrostatic pressure in the pericardial sac. The Q-T interval is usually normal.

*Constrictive pericarditis* may be recognized in the electrocardiogram by the finding in serial tracings of a rapid and steady decrease of wave amplitude in all leads together with an increase in the heart rate. T waves are frequently inverted. In the late stages of constrictive pericarditis the voltage is extremely low. Similar low voltage is seen in advanced untreated hypothyroidism where the heart rate is however very slow in contrast to that of constrictive pericarditis (Figs 5b and 23).

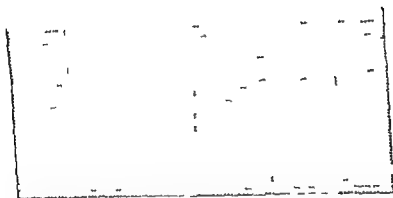


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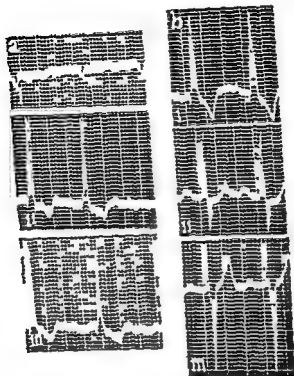


Fig 21 a Atrial fibrillation in case of 10-year-old boy with rheumatic carditis and mitral stenosis. Coarse "f" waves of fibrillation and right axis deviation are present. Digitalis effect noted by depressed and inverted T waves in leads 2 and 3.

b Standard limb leads taken from 14-year-old boy with aortic insufficiency of rheumatic origin. Conduction is delayed in the entire pathway the P-R interval being 0.24 second and the QRS 0.11 second. Marked left axis deviation is present.

effect may result from an abrupt reversal of shunt in a wide septal defect. The electrocardiogram often shows characteristic signs of right ventricular strain and hypertrophy of sudden appearance and if the patient should survive as sudden disappearance. There are deep S waves and depressed S-T segments in lead I with deep Q and elevated S-T segments in lead 3. T waves are inverted in lead 3 and over the right precordium where also R waves are often noted. The condition is most rare in a child.

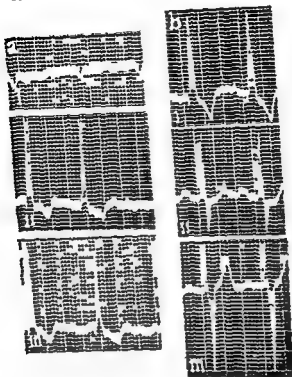


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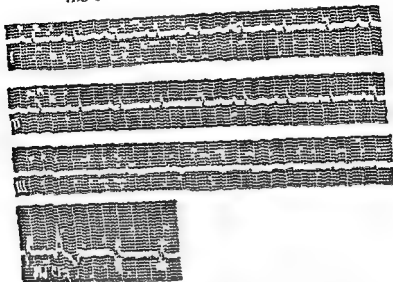


Fig 73 Standard limb lead tracing with reversed type lead CF4—seen occasionally in old editions of books on electrocardiography—from 12 year-old girl with constrictive pericarditis. There is marked low voltage. The heart rate is rapid (113 per minute). The P-R interval is 0.16 second. The Q-T interval of 0.40<sup>1</sup> second is prolonged. T waves are inverted in lead 3 and slightly biphasic in leads 1 and 2. An occasional ventricular premature contraction is present.

### Digitalis and the Electrocardiogram

The desired and usual therapeutic effect of digitalis is in slowing the heartbeat. Besides acting directly on heart muscle digitalis acts on the cardioinhibitory center and through vagal stimulation depresses the conduction of impulses between the atria and ventricles. The heart rate is lowered, the P-R interval is lengthened and the Q-T interval is shortened as the lowered level of sensitivity to calcium present in the heart in failure again becomes normal. The normal upward concavity of the S-T segment is deepened and the segment becomes depressed except occasionally in the VR lead where it may be elevated. These effects may be seen in precordial as well as in standard limb leads. Maintained digitalis effect is followed by a lowering of T wave amplitude and final inversion of the T deflection (Figs 21a, 25 and 36B).

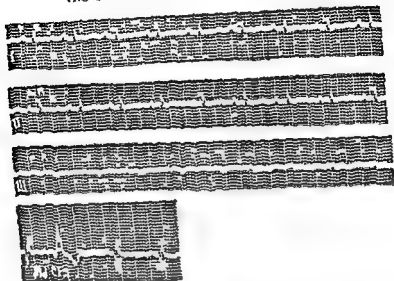


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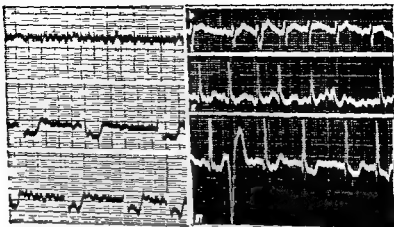


Fig 25 a b Digitalis effect seen in standard limb leads of two children receiving digitalis for heart failure

a Tracing shows slow atrial fibrillation with slight right axis deviation and coarse atrial oscillations S-T segments are deeply depressed in leads 2 and 3 From a 10-year-old boy with mitral lesions and failure

b Rapid atrial fibrillation with right axis deviation coarse atrial oscillations and frequent ventricular premature contractions The ST segments in leads 2 and 3 are depressed and concave downward Tracing of a 9 year-old boy with mitral disease

c Tracing showing digitalis effect in an adolescent boy (A F) with diagnosis of rheumatic aortic insufficiency

Sinus rhythm is present with frequent interruption by ventricular premature contractions There is electrocardiographic position of the heart The ST segments in leads 1 2, aV<sub>1</sub>, aV<sub>2</sub>, aV<sub>3</sub> and V<sub>1</sub> are depressed and concave upward

left axis deviation with semihorizontal position of the heart The ST segments in leads 1 2, aV<sub>1</sub>, aV<sub>2</sub>, aV<sub>3</sub> and V<sub>1</sub> are depressed and concave upward

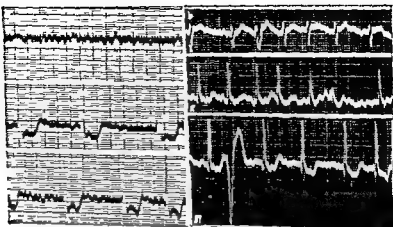


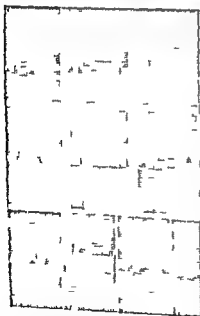
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## *The Electrocardiogram in Congenital Heart Lesions*

### **Congenital Heart Disease and the Electrocardiogram**

The electrocardiograms of many children with congenital heart lesions show no differentiating signs from those of the normal child's tracing. Similar negative findings may be seen in the electrocardiogram of the newborn infant with congenital heart disease in whom characteristic changes may appear as soon as the effect of mechanical strain becomes evident on ventricular muscle and anatomic position.

Other tracings of children with congenital heart lesions, however, fall under specific types which may aid in the diagnosis of the lesion or lesions present. Some give electrocardiographic evidence of marked positional changes owing to abnormal rotations of the heart caused by the cardiodynamics of the lesions present. Some tracings again may be typical of right or left ventricular "strain" or hypertrophy. It should be noted in this connection that ventricular hypertrophy is recognized best in the unipolar precordial leads and that deviation of the electrical axis to the right or left in the standard limb leads is frequently due to rotation of the heart and reliance on this sign as an indication of hypertrophy is often misleading. Marked right or left axis deviation is, however, usual evidence of hypertrophy of the respective ventricle. Other tracings may show diphasic QRS complexes in two or more of the standard limb leads in which the

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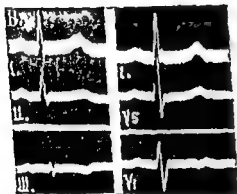


Fig 29 A Electrocardiogram of 2 year-old girl (L de R) with coarctation of the aorta (Acknowledgment to Dr Edward Hardy) The tracing shows sinus rhythm with no deviation of the electrical axis and indeterminate electrocardiograph position

B Electrocardiogram taken in E M a 14 year old girl with coarctation of the aorta and arrested sub-bacterial endarteritis and calcified mycotic aneurysm below the site of coarctation

The findings were corroborated by autopsy The electrocardiogram shows sinus rhythm with low voltage in lead 3 (The clinical findings in this case were described in *Am Heart J* 20:37 1940)

*Subaortic stenosis* Owing to a developmental arrest in the involution of the bulbus cordis a shelflike band of tissue persists below the aortic valves Electrocardiographic findings are similar to those noted above (Fig 30)

*Bicuspid aortic valve* No characteristic electrocardiographic signs are found in this lesion (Fig 31)

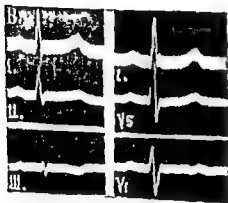


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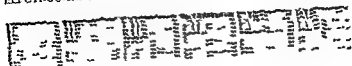


Fig 33 Tracing showing sinus rhythm with right axis deviation and heart in the vertical electrocardiogram position.

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The X

pulmonic conus

As this was referred by Dr Ruth Cudmore )

*The electrocardiogram in anomalies of the coronary arteries*  
Coronary arteries may vary in size in number in distribution and in origin. The most important clinical anomaly of the coronary system is found in those cases where the coronary artery arises from the pulmonary artery. Here signs of coronary insufficiency and myocardial ischemia appear. Patches of wedge shaped degeneration

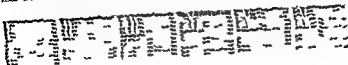


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Fig 35 Right bundle branch block is seen in this case (A F) of at al

U U E1 L K11

partial *situs inversus* the chambers of the heart are reversed A mirror image of lead I\* is present in the electrocardiogram and leads 2 and 3 are interchanged Two of the unipolar limb leads are also interchanged VR taking the character of the normal VL lead and VL resembling a usual VR lead Precordial leads are similar to those over the right chest in the normally placed heart (Figs 38 and 39)

Less commonly an isolated dextrocardia with reversal of heart chambers is seen Differentiation from the above form is made by physical and fluoroscopic examination as electrocardiographic changes described above are also present in this condition (Fig 40)

Isolated dextrocardia without reversal of heart chambers may be congenital but is commonly associated with external factors which cause a marked counterclockwise rotation of the heart on its long axis The right ventricle will then be shifted to the right and dorsal

Look into a mirror held on a stand in front of the tracing before the observer lead I will appear upright Also if the right and left arm electrodes are reversed the outline will be upright

Fig 35 Right bundle branch block is seen in this case (A F) of at al

partial situs inversus

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Fig 37 Trac of 6 months old child with severe "aortic stenosis" and "aortic murmurs" were at in this type of case. The tracing is

withally out was prominent and tortuous. The left atrium and ventricle were hypertrophied and dilated and showed extensive fibrosis. Microscopically endocardial fibrosis was marked. Subendocardial necrosis and myocardial degeneration and fibrosis were present.

of Group 3. The positional changes present however disappear on removal of the offending agent (Fig 41A and B).

*Dextrocardia combined with pulmonary stenosis* shows a mirror picture of lead I in which a large R deflection is present. R is also prominent (Fig 42).

*The electrocardiogram in uncomplicated Eisenmenger's complex.* In this not uncommon congenital heart lesion is dextroposition of the aorta with a high interventricular septal defect and dilated or

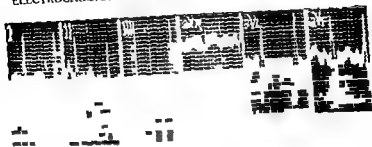


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*The electrocardiogram in pure pulmonic stenosis* Pulmonic stenosis may be of valvular type in which the three semilunar cusps are fused or it may be infundibular in type where the pulmonary conus is involved in developmental arrest and is narrowed. In either type right ventricular hypertrophy is present and usually appears in precordial leads of the electrocardiogram. Right bundle branch block is not rare in this condition (Fig. 45A and B).

*The electrocardiogram in tetralogy of Fallot* In this common combined congenital lesion of the heart pulmonic stenosis is associated with dextroposition of the aorta, ventricular septal defect and right ventricular hypertrophy. The electrocardiogram shows marked atrial and right ventricular hypertrophy. The P deflections are large and peaked especially in lead 2. Q3 is usually prominent and the T waves are often diphasic or inverted in the standard limb leads. R waves are high in  $V_1$  and VR and the S deflections are deep over  $V_5$  and  $V_6$  positions. First-degree a-v block may be seen frequently in the tetralogy while higher degrees of a-v block and bundle branch block are less common (Fig. 46A, B and C).

*The electrocardiogram in transposition of the great vessels* This anomaly is probably due to failure of normal rotation in the early stages of heart development. The pulmonary artery arises from the left ventricle and the aorta from the right ventricle. Life depends on associated lesions. The electrocardiogram shows no characteristic signs (Fig. 47).

*The electrocardiogram in tricuspid atresia* There is an occlusion or hypoplasia of the tricuspid valve of infective or developmental origin. A nonfunctioning right ventricle and hypoplastic or atretic pulmonary artery are concomitant findings in this malformation. To be compatible with life this lesion inevitably presents an associated lesion or lesions of which the most common is an atrial septal defect.

The electrocardiogram shows atrial and left ventricular hypertrophy. Left axis deviation is usually but not always present, depend

(Text continued on p. 75)

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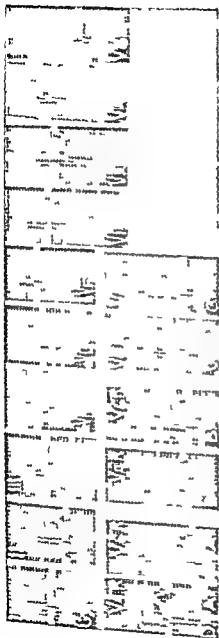
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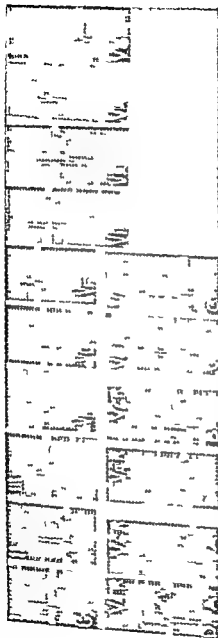
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C The same patient with arm electrodes reversed and the leg electrode attached in the right leg instead of the left one In this instance also the tracing is similar to that of B

D The true precordial leads V R and V R in this case seen in the three electrode positions A B and C noted above The three types of tracings are alike



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*The electrocardiogram in anomalous placement of tricuspid valves (Ebstein's disease)* The posterior leaflet is displaced downward into the right ventricle. Right atrial enlargement and tricuspid insufficiency are commonly present. The electrocardiogram shows right atrial hypertrophy with large and wide P waves and frequent conduction disturbances. Paroxysmal tachycardia and premature contractions may be of frequent occurrence (Fig 49)

*The electrocardiogram in isolated ventricular septal defects (maladie de Roger)* The lesion is commonly high in the septum and ventricular hypertrophy is uncommon. The electrocardiogram is usually noncontributory to the diagnosis of this lesion. Large diphasic QRS complexes with components of equal size (Katz-Wachtel sign) may occasionally be present in two standard limb leads but this sign is not pathognomonic and may disappear in the older child.

It is an interesting fact that conduction is so rarely affected in defects of the ventricular septum. Even when the septum is absent in *cor biatriatum trilobulare* or *cor bilobulare* and where the position of the bundle and its branches is most irregular, conduction is rarely disturbed. As the bundle develops before the development of the septum, it is unlikely to be affected by later appearing malformations of the septum. However, complete or incomplete a-v block does occur although rarely, and bundle branch block also may be found in defects of the ventricular septum (Fig 50).

*The electrocardiogram in single ventricle with rudimentary outlet chamber (cor biatriatum trilobulare)* There is a persistence of the common ventricle and bulbus cordis owing to an early arrest of development of the heart. One or both vessels may arise

(Text continued on p 78)

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1. Tracing showing mirror image in lead I accompanied by upright P waves in this lead. On reversal of arm leads, right becomes left axis deviation and on interchange of leads aV and aV the heart is seen to be in a semihorizontal electrocardiographic position.

The patient was a 5 month-old girl (K.L.) referred by Dr. Edward Hardy. The infant showed no cyanosis and was well nourished and developed. An isolated dextrocardia with persistent left aortic arch was present.

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Fig 41 B

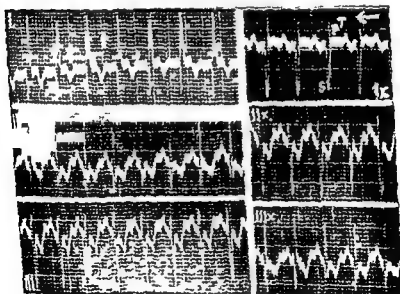


Fig 42. Tracing of 4-month-old cyanotic infant (I de C) with dextrocardia, was inversus and signs suggesting *pulmonic stenosis* as the main lesion. The standard limb leads show marked deviation of the electrical axis to the left, which on reversal of the arm terminals (x) becomes marked right axis deviation (Lead Ix should be read from right to left.)



Fig 41 B

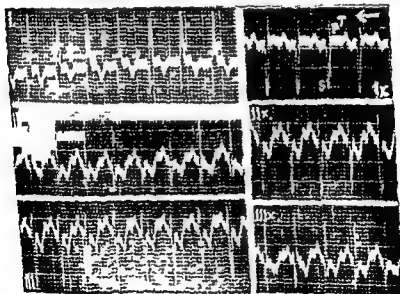


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atrial septal defect. The tracing shows evidence of right ventricular hypertrophy and large P deflections especially in lead 2 are present (Fig 52)

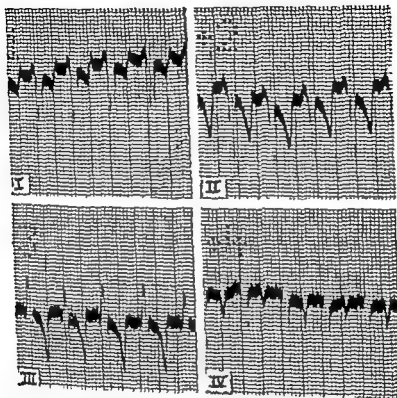


Fig 44 From an electrocardiographic tracing described by Dr Paul A. di Sant'Agnesse et al as Case # 2 (W I) in their excellent paper Glycogen Storage Disease of the Heart. *Proc. Natl. Acad. Sci. USA* 47: 1000, 1957.

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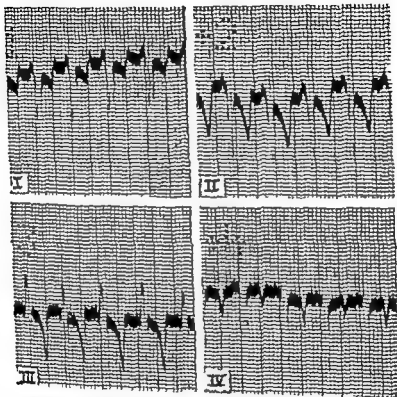


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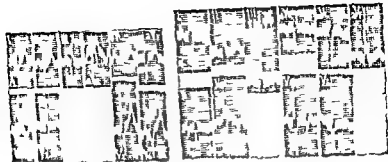


Fig 46 A Electrocardiogram of 16-month-old boy (EN) with clinical diagnosis of tetralogy of Fallot confirmed by autopsy. Cyanosis and polycythemia were present. A harsh systolic murmur was heard over the heart, loudest in the third left intercostal space. The pulmonic second sound was diminished. Under X ray examination the heart was seen to be boot shaped in the antero-posterior view with a concavity noted in the area of the pulmonary arc and the great vessels were of narrow width in this view. The lung field " " " "

B Tracing of 7 year-old boy  
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Marked right electrical axis deviation with vertical electrocardiographic po

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was removed to the left subclavian artery

Marked right electrical axis deviation with vertical electrocardiographic po-

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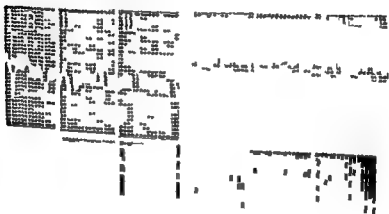
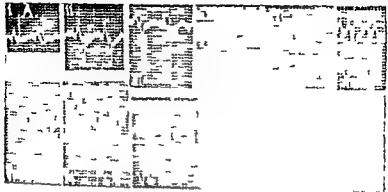


Fig 88 A Tracing taken on a 1 month-old girl (M H) who had shown moderate generalized cyanosis since birth. A harsh systolic murmur was heard loudest over the third and fourth left intercostal spaces and was transmitted over the precordium and back. A systolic thrill was palpable over the area of maximum intensity. The X ray studies showed an absence of the pulmonary arc giving a bootlike cardiac shadow.

The electrocardiogram is characteristic of right atrial and left ventricular hypertrophy. Left axis deviation with horizontal electrocardiographic position of the heart is present. P deflections are large and peaked.



Fig 11 A Tracing taken on a 9 month-old girl (MH) who had shown moderate generalized cyanosis since birth. A harsh systolic murmur was heard loudest over the third and fourth left intercostal spaces and was transmitted over the precordium and back. A systolic thrill was palpable over the area of maximum intensity. The X ray studies showed an absence of the pulmonary artery shadow giving a bootlike cardiac shadow. The electrocardiogram is characteristic of right atrial and left ventricular hypertrophy. Left axis deviation with horizontal electrocardiographic position of the heart is present. P deflections are large and peaked.



anterior oblique view no left atrial accentuation was seen on the barium filled esophagus. Repeated electrocardiograms showed as above the large and wide M waves in lead 2 typical of atrial hypertrophy. There was no deviation of the electrical axis. Arrhythmia has been a constant finding. Short phases of sinus bradycardia, at 52 beats a minute vary with runs of atrial paroxysmal tachycardia with a rate of 158 per minute. Frequently ectopic beats appear singly. Clinical diagnosis, Ebstein's disease.

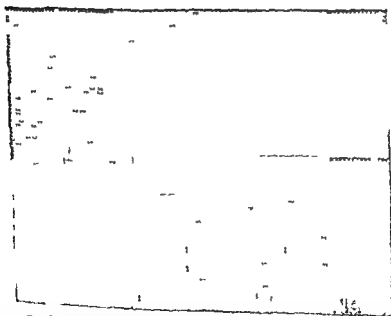


Fig. 40 Tracing from a symptom free 6 year-old boy (R O ) with ventricular septal defect who showed a harsh high pitched systolic murmur at third left intercostal space which had been noticed since birth. A systolic thrill was palpable in the same area. X ray studies showed a normal heart outline. The electrocardiogram shows sinus rhythm with no delay in conduction. T waves are large.

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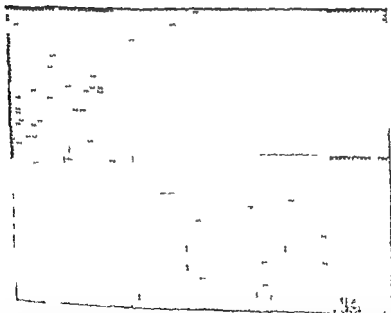


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cardiograph position. Repeatedly tracings over the precordium and around the chest were of similar form although varying in voltage.

B Tracing of 4-month-old Chinese girl (C Y) who entered the wards critically ill with pneumococcal meningitis. No heart murmurs were noted but the child was cyanotic and died soon after admission.

At autopsy a cor biatritum trilobulare was found. The heart weighed 40 gm and was triangular in shape. The aorta arose from the main chamber to the right of a small pulmonary artery which originated in a rudimentary chamber. A single atrioventricular valve was present dividing the atria from the single ventricle. A wide atrial-septal defect was present. The standard limb lead tracing shows slight right axis deviation with elevation of the S-T segment in lead 2.

Fig 51 Tracing from 2 month old boy (I R) who was admitted to the wards in critical condition with marked cyanosis and dyspnea. The heart was markedly enlarged. A harsh systolic murmur was present (although at 4 weeks neither enlargement of the heart nor cyanosis had been present). Post mortem examination showed marked hypertrophy and dilation of the right ventricle. All pulmonary veins emptied into the coronary sinus which opened into the dilated right atrium. The valve of the foramen ovale was open on the right. The tracing shows atrial and right ventricular hypertrophy. Marked right axis deviation with vertical electrocardiograph heart position is present. The P deflections are high and wide especially so in lead 2. There is of high amplitude in aV

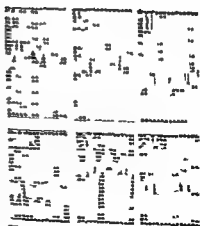


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brain lesions especially those affecting the medulla. It may be noted in hypothyroidism. Sinus bradycardia is rare in the cardiac child except as a digitalis effect. Sinus arrhythmia and sinus arrest are usual accompaniments of a slow heart rate (Fig 53c).

### Sinus Arrhythmia

Sinus arrhythmia is an irregularity of sinus rhythm in which the R-R cycles vary more than 0.1 second. The arrhythmia has no clinical significance and is common in the child especially so in those over 7 years of age (Fig 53d).

### Sinus Arrest

Sinus arrest is an arrest of sinus rhythm in which a complete cardiac cycle is blocked or fails to appear. The resultant pause almost equals two normal cycles. The condition may be physiological and is not rare in the normal child (Fig 53e).

### Premature Contraction

An abnormal heart contraction that arises from an ectopic focus and disturbs the regularity of the basic rhythm is a premature contraction. Premature contractions may be of atrial, atrioventricular, nodal or ventricular origin. They may be recognized in the electrocardiogram both by their abnormal position in the cycle and by their bizarre form. A compensatory pause may be seen following the ectopic systole. There is no compensatory pause when the ectopic beat is interpolated. In a slow rhythm the prematurely contracted ventricle has then had time in which to recover from its refractory period and is receptive for the arrival of the normal excitation impulse (Fig 16). Premature contractions of each type may be found in the normal infant and child although those of atrial form are the most usual. They are also found in acute rheumatic fever and other infectious diseases and in typhoid fever during the period of slow rate. Premature contractions may also be present in congenital heart disease as in atrial and tricuspid valve anomalies although this finding is not as frequent as it is in the adult with an atrial septal defect.

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*Coupling* or alternation of ectopic with regular contractions is not rare in children especially in those who have had recent pulmonary infections and during digitalis toxicity (Fig 54b and c)

*Atrial premature contractions* Ectopic P waves are usually inverted they appear prematurely and are followed by a P R interval that is not shortened Atrial premature contractions may be present in the normal child They may also appear in acute rheumatic fever and other infections They are occasionally seen in the child with congenital heart disease (Fig 54a and b)

*Nodal premature contractions* arise in the atrioventricular node and cause the P deflection by retrograde action to appear just before during or after the QRS complex which is of supraventricular type Nodal systoles may be found in the normal child's tracing as well as in those with acute rheumatic fever and other infections (Fig 54c)

*Ventricular premature contractions* arise in ectopic foci of the ventricle The QRS complex so formed is bizarre in outline resembling somewhat the wide notched QRS curve of a blocked bundle branch The contraction may be of right or of left ventricular origin and may be recognized as such in the standard lead I If the main QRS deflection in lead I is upright the systole is of right ventricular origin if downward of left ventricular origin

Although less common in the normal child than premature systoles of supraventricular origin ventricular premature contractions do occur in the child without cardiac pathology They are more frequent in children with acute infections especially of rheumatic and pulmonary etiology and in nephritic and endocrine disorders

a Sinus tachycardia with rate of 200 per minute in 8 month-old male with acute infection Atrial and ventricular complexes and intervals are of normal outline

b Same in 8 year-old girl with her heart

c Sinus bradycardia in 12 year-old boy Heart rate 100 complex Low U

d Sinus arrest in 12 year-old girl with joint pains The P P intervals vary in length

e Sinus arrest in normal 6 year-old child There is a pause almost equal to two normal beats in which the heart waits to receive stimulus from the normal pacemaker

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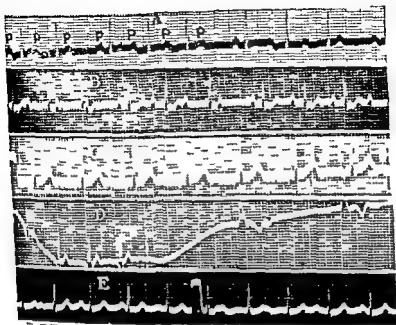


Fig 55 A, B Two cases showing Wenckebach's syndrome in 6- and 8 year old boys, respectively suffering with acute rheumatic carditis. The P R intervals increase in duration progressively until the P wave falls during a refractory period of the ventricle and is blocked. The following P R interval is of normal duration and the cycle is repeated.

C Atrioventricular or nodal rhythm in a 6-year-old normal boy with acute upper respiratory infection. T waves are inverted, and the P R interval is shortened. U waves are visible following the T deflections.

D Paroxysmal a.v. rhythm in 8 year-old girl showing cardiac asystole and occasional ventricular escape beat during ocular pressure. (Case of Dr Alfred Langemann.)

E AV interference dissociation appearing in an 11 year-old boy with rheumatic carditis. (Case of Dr Herbert von Gal.) The atria responding to the depressed S-A node beat at a slower rate than that of the ventricles which react to an ectopic focus in the a.v. node or ventricle.

A V rhythm may be present as a congenital anomaly of conduction and is usually of little clinical significance. The rhythm is often transitory and the position of the P wave may vary in respect to the QRS complex especially in those cases where a.v. rhythm is due to distal or other toxic effect (Fig 55C and D).

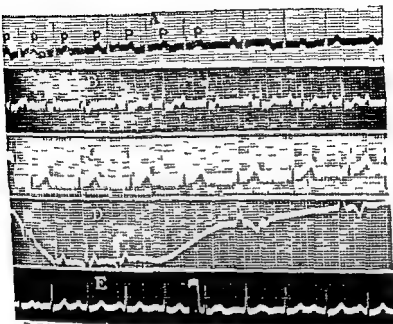


Fig 55 A, B Two cases showing Wenckebach's syndrome in 6- and 8 year old boys, respectively suffering with acute rheumatic carditis. The P-R intervals increase in duration progressively until the P wave falls during a refractory period of the ventricle and is blocked. The following P-R interval is of normal duration and the cycle is repeated.

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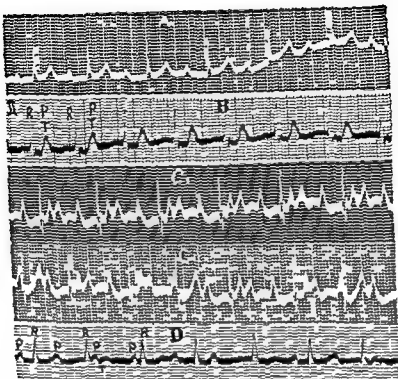


Fig 56 A First-degree a v block of transitory duration. The P waves are notched and widened. The PR interval is 0.32 second. The PR segment is lowered (Case of acute rheumatic fever).

B As in A. P waves are superimposed on T deflections in 6 year-old child with a rheumatic fever.

C Second-degree a v block. P and R waves fall regularly and PR intervals are equal. The first atrial or P wave in each cycle is blocked by a refractory ventricle giving a 2:1 degree of block. Note the Ta wave (inverted) following the second P deflections.

D The same child as above during phase of 1:1 a v block. Three year-old boy with tetralogy of Fallot. The S-T segment is occasionally depressed by a Ta wave.

E Complete a v block in an 11 year-old boy with ventricular septal defect. P waves and R waves fall regularly but bear no relationship to one another. The PR intervals are irregular.

node during its refractile stage when the ventricular response being blocked, no QRS complex will appear. The succeeding P deflection is followed by a normal PR interval and the cycle of increasing a v conduction is repeated (Fig 55A and B).

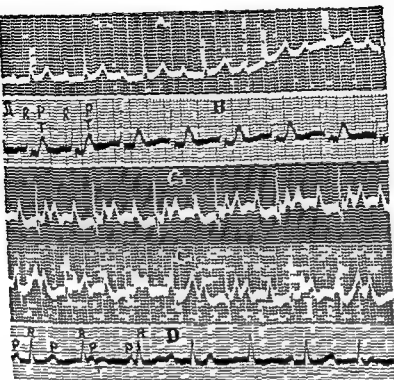


Fig 56 A First-degree a v block of transitory duration The P waves are notched and widened The PR interval is 0.3 second The PR segment is lowered (Case of acute rheumatic fever)

B As in A P waves are superimposed on T deflections in 6 year-old child with a live carditis

C Second-degree a v block P and R waves fall regularly and PR intervals are equal The first atrial or P wave in each cycle is blocked by a refractory ventricle giving a 2 degree of block Note the Ta wave (inverted) following the second P deflections

D The same child as above during phase of 1:1 a v block Three year-old boy with tetralogy of Fallot The S-T segment is occasionally depressed by a Ta wave

E Complete a v block in an 11 year-old boy with ventricular septal defect P waves and R waves fall regularly but bear no relationship to one another The PR intervals are irregular

node during its refractile stage when the ventricular response being blocked no QRS complex will appear The succeeding P deflection is followed by a normal PR interval and the cycle of increasing a v conduction is repeated (Fig 55A and B)

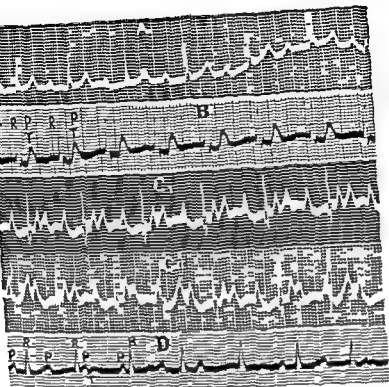


Fig. 56 A First-degree a v block of transitory duration The P waves are notched and widened The P R interval is 0.32 second The P R segment is lowered (Case of acute rheumatic fever)

B As in A P waves are superimposed on T deflections in 6 year-old child with active carditis

C Second-degree a v block, P and R waves fall regularly and P R intervals are equal The first atrial or P wave in each cycle is blocked by a retrograde Ta wave (inverted)

2 1 1 1 a v block Three segment is occasionally de

viewed by a Ta wave

D Complete a v block in an 11 year-old boy with ventricular septal defect P waves and R waves fall regularly but bear no relationship to one another The P R intervals are irregular

node during its refractile stage when the ventricular response being blocked no QRS complex will appear The succeeding P deflection is followed by a normal P R interval and the cycle of increasing a v conduction is repeated (Fig 55A and B)

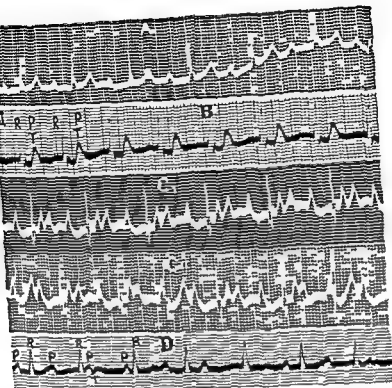


Fig. 56 A First-degree a-v block of transitory duration. The P waves are notched and widened. The P-R interval is 0.3 sec. second The P-R segment is lowered (Case of acute rheumatic fever)

B As in A. P waves are superimposed on T deflections in 6 year-old child with active carditis

C1 Second-degree a-v block, P and R waves fall regularly and P-R intervals are equal. The first atrial or P wave in each cycle is blocked by a re-verse the Ta wave (inverted)

2111 a-v block. Three segment is occasionally de-

pressed by a Ta wave

D Complete a-v block in an 11 year-old boy with ventricular septal defect. P waves and R waves fall regularly but bear no relationship to one another. The P-R intervals are irregular

node during its refractile stage when the ventricular response being blocked no QRS complex will appear. The succeeding P deflection is followed by a normal P-R interval and the cycle of increasing a-v conduction is repeated (Fig. 55A and B)

or ventricle beat at a more rapid rate QRS complexes appear more frequently and usually without relation to P waves however, normal conduction may appear for one or more cycles in the course of the rhythm (Fig. 55E)

### Atrial Paroxysmal Tachycardia

This is an abnormal transitory rhythm consisting of a phase of rapid atrial premature contractions which appear without pause regularly one after another The condition is occasionally seen in the newborn in whom no clinical evidence of cardiac disease can be found It may also be found in children with congenital heart disease especially in those with atrial and tricuspid leaflet anomalies in whom it may appear on effort or during acute infections It may occur in cases of short P R interval or Wolff Parkinson White syndrome It is not common in acute rheumatic fever although it may occur (Figs 26 49 [L2 and 3] and 57C)

### Atrioventricular Nodal Paroxysmal Tachycardia

This is an ectopic regular rapid rhythm with its origin in the a v node P waves may precede or fall upon or follow the supraventricular type of QRS complexes which are dominant The tachycardia may appear in a normal child and no cause may be found It may be seen in children with congenital heart disease and in those with short P R conduction time (Figs 55D and 57D)

### Ventricular Paroxysmal Tachycardia

This is an abnormal tachycardia which originates in the ventricle and consists of a succession of rapid and almost regular ectopic ventricular beats The rhythm is very rare in the child especially in those under 10 years of age It may appear in the rheumatic cardiac with widespread myocardial damage and rarely in congenital cardiacs (Fig. 57E)

### Atrial Flutter

Atrial flutter is a disorder of atrial conduction in which there are abnormal rapid and regular atrial contractions with a rate averaging

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common in atrial flutter seen in infancy. Various degrees of block may be present in the same patient.

The condition is rare in the child although many of the tachycardias found in the newborn are due to this condition and it may occasionally occur in the child with advanced mitral disease or in cases with hypertrophy and dilatation of the right atrium as in Ebstein's disease.

The electrocardiogram shows toothlike regular atrial flutter waves which follow one another without pause and without respect to the ventricular responses which fall regularly usually with a 1:2, 1:3 or 1:4 or mixed degree of block. Flutter or *f* waves are best seen in leads 2 and 3 and in  $V_1$  and  $V_4$  (Fig. 57B).

### Atrial Fibrillation

Atrial fibrillation is a completely irregular heart action in which the atria are in a constant state of rapid, irregular, and ineffectual motion and in which ventricular response is also irregular in force and rhythm. Atrial activity is represented in the tracing by unequally sized and spaced small *f* waves. P waves are absent and the QRS complexes vary in voltage and appearance (Figs. 21, 25 and 57A).

Atrial fibrillation is rarely seen in the child. It may be present in severe rheumatic carditis especially in those children who have greatly enlarged hearts and mitral lesions. The rhythm may be paroxysmal or transitory in type and may appear as a sign of digitalis toxicity when it will disappear on the removal of the drug. It may also occur as a transient finding in typhoid fever and diphtheria and has been found on embolization of a coronary artery from vegetative endocarditis.

### Ventricular Fibrillation

Fibrillation of the ventricles is a grave disorder of the heartbeat in which the ventricles contract with total irregularity of force and rate. The electrocardiogram shows large, rapid, irregular oscillations of

common in atrial flutter seen in infancy. Various degrees of block may be present in the same patient.

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### Ventricular Fibrillation

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The electrocardiogram shows large rapid irregular oscillations of

## Appendix

### Boys

Age	Number of Cases	Average	$\pm$ Standard Deviation	Range
3	9	0.13	0.04	0.12-0.15
4	18	0.13	0.03	0.10-0.16
5	24	0.14	0.02	0.12-0.17
6	29	0.13	0.02	0.11-0.17
7	33	0.14	0.02	0.11-0.19
8	31	0.14	0.02	0.10-0.18
9	33	0.13	0.01	0.10-0.16
10	27	0.14	0.01	0.10-0.18
11	19	0.14	0.01	0.12-0.16
12	13	0.14	0.04	0.12-0.18

### Girls

Age	Number of Cases	Average	$\pm$ Standard Deviation	Range
3				
4	22	0.13	0.01	0.10-0.16
5	33	0.12	0.02	0.10-0.16
6	29	0.13	0.01	0.12-0.16
7	35	0.14	0.02	0.11-0.16
8	30	0.14	0.01	0.12-0.16
9	6	0.14	0.02	0.12-0.16
10	18	0.14	0.02	0.12-0.16
11	21	0.14	0.02	0.12-0.16
12	9	0.14	0.02	0.12-0.16

Table 8 Showing average P R intervals without reference to heart rate found in a study of 77 normal school children 3 to 13 years of age. (Student's t-test)

## Appendix

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11	21	0.14	0.02	0.12-0.16
12	9	0.14	0.02	0.12-0.16

Table 8 Showing average R intervals without reference to heart rate found in a study of 77 normal school children 3 to 13 years of age

Cycle length	Square root
$\sqrt{0.44}$ ec	0.6635
0.46	0.678
0.48	0.692
0.50	0.707
0.5	0.722
0.53	0.727
0.54	0.735
0.56	0.749
0.58	0.760
0.60	0.775
0.62	0.787
0.64	0.800
0.66	0.812
0.68	0.825
0.70	0.835
0.72	0.855
0.74	0.866
0.76	0.870
0.78	0.882
0.80	0.895
0.82	0.905
0.84	0.915
0.86	0.927
0.88	0.935
0.90	0.950
1.1	1.06
1.16	1.08

Table 11 Approximate square roots of cycle lengths for use in determining  $QT_c$  values

### Einthoven's Triangle

In an electrical sense the right shoulder the left shoulder and the left hip form an equilateral triangle in the frontal plane around the heart. The heart thus lies approximately equidistant from each of the three points in the relatively homogeneous substance of the body. The complicated pathway of electrical activity passing through the heart on atrial or ventricular activation may be reduced to a single vector which may be calculated from any two of the three standard leads by Einthoven's formula\* or simply by using Einthoven's triangle which is a more crude but useful method (Fig. 59).

Tangent of alpha  $= \frac{e - c}{c \sqrt{3}}$  where alpha equals the angle between the axis and horizontal  $e$  the amplitude in millimeters of the QRS wave in lead 1 and  $c$  that of QRS in lead 3. The length of the axis or the manifest potential difference is calculated from  $\frac{E \approx e_1}{\cos (a-60^\circ)}$

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0.86	0.927
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Table II Approximate square roots of cycle lengths for use in determining  $QT_0$  values

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Tangent of alpha  $= \frac{e - e_2}{e \sqrt{3}}$  where alpha equals the angle between the axis and horizontal  $e$  the amplitude in millimeters of the QRS wave in lead I and  $e_2$  that of QRS in lead II. The length of the axis or the manifest potential difference is calculated from 
$$\frac{E \approx e_2}{\cos (2-60^\circ)}$$

amplitudes in millimeters of  $R_3$  and  $S_1$  from the sum of  $R_1$  and  $S_3$   
W D White suggests that if this index measures over 30 mm abnormal  
left axis deviation is present with normal border line of 20 to 30  
if it measures under  $-15$  mm abnormal right axis deviation is  
present with a normal border line of  $-10$  to  $-15$

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